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The association between prenatal cocaine exposure and physiological regulation at 13 months of age

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Abstract

Background—This study examined the association between prenatal cocaine exposure (PCE) and autonomic regulation at 13 months of age.

Methods—Measures of respiratory sinus arrhythmia (RSA) were obtained from 156 (79 exposed, and 77 nonexposed) infants during baseline and during tasks designed to elicit positive (PA) and negative affect (NA).

Results—There was a significant suppression of RSA during the negative affect task for nonexposed infants but not for exposed infants. Maternal symptoms of depression or anxiety (MDA) did not mediate this association. However, gender and MDA did moderate this association such that exposed boys and exposed infants whose mothers had higher levels of MDA had an increase in RSA during a task designed to elicit NA rather than the typical pattern of RSA suppression.

Conclusions—These results suggest that there are several possible pathways from PCE to physiological dysregulation during late infancy.

Keywords

Autonomic regulation; prenatal cocaine exposure; respiratory sinus arrhythmia; maternal depression/anxiety; gender differences

Prenatal exposure to cocaine (PCE) has been linked to nonoptimal developmental outcomes including an increased risk of dysregulation during infancy and childhood (Coles, Platzman, Smith, James, & Falek, 1992; Karmel & Gardner, 1996; Mayes & Bornstein, 1995). One particularly useful biological measure of regulatory processes is respiratory sinus arrhythmia (RSA), which Porges terms vagal tone (Vna; Porges & Byrne, 1992). RSA is a measure of the variability in heart rate (HR) that occurs at the frequency of respiration and is believed to index the parasympathetic influence of heart rate variability (HRV) via the vagus nerve. Two commonly used indices of physiological regulation (Porges, 1996) include baseline RSA (BRSA) and changes in RSA during environmental demands (RSA regulation; Bornstein & Suess, 2000; Calkins, 1997). BRSA is a measure of the infant's ability to maintain physiological homeostasis during periods of minimal external stimulation. During challenges to homeostasis as a result of exogenous stimulation, the myelinated vagal system optimally responds by functioning as a brake (Porges, Doussard-Roosevelt, Portales, &

Greenspan, 1996) and decreasing RSA (suppression). RSA suppression during periods of environmental challenge is believed to reflect the infant's ability to appropriately engage or disengage with the environment (Bornstein & Suess, 2000; Porges, 1996). Thus, the measurement of change in RSA from baseline to challenging situations is an important concurrent and predictive index of parasympathetic regulation in infants.

The majority of studies examining parasympathetic regulation as a function of PCE have focused on neonates and findings have been somewhat mixed. Some studies suggest increased parasympathetic activity during rest among CE neonates (Regalado, Schechtman, Khoo, & Bean, 2001; Silvestri, Long, Weese-Mayer, & Barkov, 1991), while others have not found any effects of PCE on BRSA (Mehta et al., 1993), perhaps due to lower sample sizes. Beyond the neonatal period, baseline indices of RSA are not as indicative of an infant's ability to modulate arousal (DiPietro, Porges, & Uhly, 1992). However, few studies have examined changes in physiological regulation in response to environmental challenge among CE infants. One exception is findings from the sample in the present study at 7 months of age indicating that CE infants did not have a significant RSA suppression during a task designed to elicit NA at 7 months although the NE infants did show the expected RSA suppression (Schuetze, Eiden, & Coles, 2007). It is unclear if this difference persists into later infancy. Thus, the first goal of this study was to examine the association between PCE and physiological regulation during environmental stimulation at 13 months of age. We hypothesized that CE infants would have a lower BRSA, reflecting a less optimal capacity to react appropriately to environmental stimulation, and a reduced RSA suppression during environmental challenge, indicating poorer physiological regulation.

Viewing developmental trajectories from a developmental psychopathology framework suggests that there may be multiple pathways to risk among biologically vulnerable children (Luthar & Zelazo, 2003). One such pathway for CE infants may be through higher maternal depression/anxiety (MDA). Numerous studies have found that mothers who used cocaine during pregnancy reported higher levels of depression and anxiety (Singer et al., 1995; Woods, Eyler, Behnke, & Conlon, 1993). There is a large literature highlighting the predictive role of MDA for numerous developmental outcomes, including regulation (Davis et al., 2007; McGrath, Records, & Rice, 2008). Infants of depressed mothers have elevated cortisol and lower vagal tone reflecting autonomic dysregulation (Feldman & Eidelman, 2007). Thus, MDA may be one pathway explaining the potential association between PCE and dysregulation among exposed infants. Consequently, another goal of the present study was to examine if MDA mediates or moderates the association between PCE and infant regulation. For moderation, we hypothesized that the association between CE and regulation would be stronger under conditions of increased MDA.

Evidence is also accumulating that suggests gender may moderate the association between PCE and developmental outcome. Studies have consistently found that boys are more vulnerable to a range of developmental problems (Gualtieri & Hicks, 1985) including externalizing behavior problems (Campbell, Shaw, & Gilliom, 2000). Gender differences seem particularly pronounced among at-risk infants (e.g., Hay, 1997; Weinberg, Tronick, Cohn, & Olson, 1999) including CE infants (Bendersky et al., 2006; Moe & Slining, 2001). Consequently, another goal of the present study was to determine if gender moderated the association between CE and physiological regulation. We hypothesized that the association between CE and regulation would be stronger for boys.

Finally, it is important to note that cocaine is a polydrug issue. The majority of women using cocaine also use alcohol, cigarettes, and marijuana. Alcohol and cigarettes have been known to have similar effects on infant regulation (e.g., Fried & Makin, 1987; Schuetze & Eiden, 2006). Thus, the associations between CE and infant regulation can only be examined in the

context of associations with other substance use. Thus, we considered the possible impact of prenatal exposure to other substances in all analyses.

Method

Participants

Mother–infant dyads were recruited postpartum from two local hospitals into a longitudinal study of maternal substance use and child development. Of the 220 infants recruited into the study, 20 did not show up for the 13-month assessment after repeated reschedules (11 CE), 9 were unable to be located (5 CE), 14 (6 CE) were dropped (severe medical problems) or withdrew from the study, and 4 exposed infants had foster parents who were uninterested in participating. An additional 16 infants did not have complete physiological data due to equipment failure ($n = 6$), research assistant error ($n = 2$), infant irritability ($n = 5$; 2 CE) or excessive movement artifact in the EKG data ($n = 3$). One additional family was excluded from analyses because it was a multivariate outlier (cocaine, alcohol, cigarette use and gestational age were more than three standard deviations from the sample mean). Thus, the final sample was 156 (79 CE, 77 NE) dyads. There were no significant differences between families with complete versus missing data at 13 months on demographic or substance use variables. It is important to note that, although the sample size was similar to the sample size in findings reported at 7 months of age (Schuetze et al., 2007), both samples consist of a subset (those with completed physiological data at the time point of interest) of the total study sample. Only 66 CE and 65 NE infants were included in both samples. Thus, there are some differences in sample characteristics between the two reports.

Mothers ranged in age from 18 to 42 ($M = 29.69$, $SD = 5.95$); 55.3% of mothers were African-American, 12.9% were Caucasian, and 5.6% were Hispanic; 81% were receiving Temporary Assistance for Needy Families and 90.3% were single. Women were classified as either cocaine-users or ‘abstainers’. The two groups were matched on maternal education, age, race/ethnicity and infant gender; 51.9% of the infants were male (CE: 53.2%; NE: 50.6%). Gender did not differ between groups, $X^2(1) = .11$, $p > .05$. CE infants (7%, range = 33–41 weeks) were not significantly more likely to have been preterm than NE (4%, range = 36–42 weeks) infants, $X^2(1) = 10.1$, $p > .05$. All testing was conducted after age corrected for prematurity. Infants ranged from 1531 to 5072 grams at birth ($M = 3142.01$, $SD = 567.33$). Additional exclusionary criteria were: maternal age under 18 years, and prenatal illicit substance use other than cocaine or marijuana.

The study received approval from the institutional review boards of the hospitals and primary institutions of the authors. Participants received \$35.00 and \$80.00 in monetary incentives at the 1-month and 13-month visits, respectively.

Procedure

All mothers were screened after delivery for eligibility and matching criteria. Informed written consent was obtained from interested, eligible mothers. Assessments were conducted at 1 and 13 months of infant age. In the circumstance of a custody change, the legal guardian was asked to participate. Biological mothers were interviewed to obtain accurate information about prenatal substance use.

Assessment of growth and risk status—Three growth measures were taken by obstetric nurses: birthweight (gm), birthlength (cm), and head circumference (cm). Medical chart review was used to complete the Obstetrical Complications Scale (OCS; Littman & Parmelee, 1978) to assess perinatal risk factors. Higher numbers are more optimal.

Identification of substance use

Cocaine status was determined through maternal self-report, chart review which provided results of urine toxicology, and maternal hair analysis. Urine toxicologies were routinely conducted by participating hospitals. Self-reports of maternal substance use before, during and after pregnancy were obtained using the Timeline Follow-back Interview administered to the biological mother (TLFB; Sobell, Sobell, Klajner, Pavan, & Basian, 1986) at the 1-month visit. The TLFB yielded data about the average number of days per week cocaine was used, the average number of joints of marijuana, the average number of cigarettes (tobacco), and the average number of standard drinks consumed per week, as well as the mean standard drinks per drinking day and number of alcohol binges (5 or more standard drinks) during pregnancy and postpartum.

Urine toxicologies consisted of standard screening for drug levels/metabolites of cocaine, opiates, benzodiazepines, and tetrahydrocannabinol. Urine was rated positive if the quantity of drug/metabolite was >300 g/ml. Hair samples were collected from all mothers at the 1-month visit and sent to the Psychomedics Corporation for radioimmunoanalyses (RIAH). Hair samples were screened for cocaine followed by a gas chromatography/mass spectrometry (GC/MS) confirmation for positive cocaine screens.

Of the mothers in the cocaine group, 46% ($n = 36$) had positive urine toxicologies at delivery, 61% ($n = 48$) had hair samples that tested positive for cocaine during pregnancy, and 84% ($n = 66$) admitted having used cocaine in the brief self-report screening instrument administered after delivery. The majority of mothers in the cocaine group met multiple criteria for inclusion into the cocaine group. Mothers in the comparison group reported not having used any illicit substances other than marijuana and did not test positive for cocaine or other illicit substances other than marijuana on any biomarker.

Assessment of infant reactivity and regulation

Physiological regulation was assessed during a 3-minute baseline period (video), a 2-minute positive affect (PA) paradigm, a 3-minute inter-task interval (video) and a 2-minute negative affect (NA) episode by examiners blind to group status. Mothers were asked not to interact with their infant unless specifically instructed to do so. Infants were tested while seated in a high-chair. Recording began once the infant was observed to be in a stable, quiet, alert state which was induced by having the infant watch a 3-minute segment of a neutral videotape, 'Baby Einstein' (see Calkins, 1997). The PA paradigm consisted of a puppet show that measured PA in response to social stimulation using a standardized presentation (Goldsmith & Rothbart, 1999). The NA paradigm consisted of a gentle arm-restraint episode which is a widely used, well-validated measure of anger/frustration used to assess infant regulation and reactivity (Goldsmith & Rothbart, 1999; Stifter & Braungart, 1995).

A five-channel Bioamp (James Long Company, Caroga Lake, NY) recorded respiration and electrocardiograph (ECG) data. Disposable electrodes were triangulated on the infant's chest. A respiration bellows was placed at the level of the xyphoid process to measure inspiration and expiration. IBI Analysis software (James Long Company, Caroga Lake, NY) was used to process the heartrate (HR) data and to calculate respiratory sinus arrhythmia (RSA). The software synchronizes with respiration and is relatively insensitive to arrhythmia due to tonic shifts in HR, thermoregulation, and baroreceptor.

Average RSA was calculated for the 3-minute baseline, the PA task, and the NA task. Two RSA change scores, from baseline to the PA task and from baseline to the NA task, were calculated to assess physiological regulation. These were termed RSA regulation to PA (RSA-PA) and RSA regulation to NA (RSA-NA). Negative scores indicate a decrease in RSA and reflect more optimal parasympathetic regulation.

Assessment of maternal depression/anxiety (MDA)

The Brief Symptom Inventory (BSI; Derogatis, 1993), with well-established psychometrics, was used to assess MDA at 13 months. A composite variable termed MDA was created by taking the maximum score from the Anxiety and Depression subscales.

Results

Descriptive statistics

Multivariate analyses of variance (MANOVA) were conducted to examine group differences for the demographic, maternal substance use, and infant risk status variables for the exposure groups (see Table 1 for univariate results). Results indicated a significant multivariate effect of group status for prenatal substance use, $F(5, 150) = 8.16, p < .001, \eta^2_p = .22$, demographics, $F(4, 151) = 4.21, p < .01, \eta^2_p = .10$, infant risk status, $F(3, 152) = 12.46, p < .001, \eta^2_p = .19$, and postnatal substance use, $F(4, 151) = 4.14, p < .01, \eta^2_p = .10$.

Relations between study variables

We then examined the association between the prenatal and postnatal substance use, infant risk characteristics, MDA and RSA variables. Higher prenatal cocaine, $r = .25, p < .01$ and alcohol exposure, $r = .19, p < .05$, postnatal alcohol use, $r = .19, p < .05$, maternal age, $r = .17, p < .05$, parity, $r = .225, p < .01$, and MDA, $r = .18, p < .05$, were associated with RSA-NA. Scores on the OCS, $r = .17, p < .01$, were associated with BRSA. In addition, maternal education, $r = -.18, p < .05$, pregnancy cocaine use, $r = .18, p < .05$, postnatal cigarette, $r = .26, p < .001$, and alcohol use, $r = .22, p < .01$, were associated with MDA.

Group differences in RSA

Because group differences in outcome measures have been found between exposed infants in foster care and exposed infants in the custody of their biological mother (e.g., Brown et al., 2004), we conducted analyses to see if there were differences in RSA for CE infants whose primary caregiver was the biological mother ($n = 62$) as compared to CE infants who were in the care of someone other than the biological mother ($n = 17$). Separate ANCOVAs with caregiving status as the independent variable and RSA variables as the dependent variables were conducted. Maternal alcohol and cigarette use during pregnancy, maternal postnatal alcohol and cigarette use, maternal age, parity, scores on the OCS, and GA were used as covariates. There were no significant differences between infants in foster care and infants in the care of their biological mother for BRSA, $F(1,62) = 1.27, p > .05$, RSA-NA, $F(1,62) = .98, p > .05$, or for RSA-PA, $F(1,62) = 1.73, p > .05$. Consequently, foster care was not considered further, analytically.

A repeated-measures ANCOVA with condition (baseline, PA, and NA) as the within-subject factor, and group status (exposure, control) as the between-subjects factor was conducted for RSA (see Figure 1). Results indicated a significant interaction effect of group by condition, $F(4, 138) = 4.91, p < .001, \eta^2_p = .16$. CE infants had a significantly lower BRSA and significantly higher RSA during the NA task.

Group differences in RSA suppression in response to environmental challenge

Because physiological responses may be influenced by the Law of Initial Value (Wilder, 1956), the association between BRSA and subsequent RSA values was examined. BRSA was associated with both RSANA, $r = -.43, p < .001$, and RSA-PA, $r = -.67, p < .001$. Thus, measures of RSA were adjusted for baseline levels by including BRSA as a covariate in all analyses of RSA change.

ANCOVAs were conducted to look at group differences in these change scores. Results showed that CE infants had a slight increase in RSA during the NA ($M = -.012$, $SD = .018$) task while NE infants ($M = .006$, $SD = .032$) had a decrease in RSA during the NA, $F(1, 146) = 11.23$, $p < .001$, $\eta^2_p = .07$. There was no significant group difference for change in RSA during the PA task (NE: $M = -.012$, $SD = .035$; CE: $M = -.0001$, $SD = .025$), $F(1, 146) = 1.2$, $p > .05$, $\eta^2_p = .01$.

Mediational analyses

Next, we examined if there was an indirect association between PCE and autonomic regulation via MDA. CE infants had mothers with higher MDA, *Unstandardized Beta* = .004, $p < .05$. In the next step, the association between MDA and the RSA variables was estimated. MDA was not significantly associated with any of the RSA measures and, consequently, was not considered further as a potential mediator.

Moderational analyses

We then conducted analyses to examine if the association between PCE and autonomic reactivity (BRSA) and regulation (RSA-PA, RSA-NA) was moderated by infant gender. As described above, covariates were variables that were associated ($p < .10$) with either CE or RSA variables. In addition, BRSA was included as a covariate in the analyses of RSA regulation. The covariates were entered in the first step, followed by PCE and gender in the second step and the interaction term in the third step. There was no significant interaction term in the analysis for BSA, $F(1,145) = 1.62$, indicating that gender did not moderate the association between CE and BRSA. However, there was a significant interaction term for RSA-PA, $F(1,145) = 3.86$, $p < .05$, $\eta^2_p = .03$, and RSA-NA, $F(1,145) = 6.05$, $p < .05$, $\eta^2_p = .05$, indicating a significant moderation by gender for RSA regulation during environmental challenge (see Figures 2 and 3). CE boys had an increase in RSA during the PA and NA tasks rather than the RSA suppression shown by exposed girls and nonexposed boys and girls.

The same procedure was used to examine MDA as a potential moderator. Because MDA did not differ between mothers of boys and girls, $F(1,155) = .41$, $p > .05$, gender was not included as a covariate in these analyses. The median of MDA (2.33) was used to categorize MDA as low or high. There were no significant interaction terms for MDA variables in the analyses for BRSA or RSA-PA. However, MDA was a significant moderator of the association between prenatal exposure to cocaine and RSA-NA, $F(1,146) = 3.83$, $p < .05$, $\eta^2_p = .03$ (see Figure 4). Exposed infants who had mothers with higher levels of MDA had a significantly higher RSA-NA relative to exposed infants whose mothers had low MDA or unexposed infants.

Discussion

A growing body of research has found that CE infants have a higher level of problems with behavioral regulation. Several studies have indicated that this dysregulation extends to physiological measures of regulation such as lower HR (Silvestri et al., 1991), greater HRV (Regalado et al., 2001), lower BRSA and absence of RSA suppression during NA (Schuetze et al., 2007) during early infancy. Because less is known about physiological regulation in this population beyond early infancy, one goal of this study was to examine the relation between PCE and measures of physiological regulation at 13 months. Similar to previous studies that found differences in BRSA and other measures of HRV during early infancy (Regalado et al., 2001; Schuetze & Eiden, 2006; Schuetze et al., 2007), we found an association between PCE and BRSA. Infants with higher BRSA have increased positive and negative emotional reactivity during environmental stimulation (Stifter & Fox, 1990) and

exhibit increased self-soothing (Fox, 1989). Thus, our findings of lower BRSA for exposed infants suggest decreased capacity for optimal autonomic reactivity and may predict behavioral dysregulation. However, our baseline measure occurred after infants had been introduced to a new setting by novel research assistants, buckled into a high chair, and connected to physiological data acquisition equipment, all of which may have been stressors. Thus, this was not a true baseline measure obtained in the absence of environmental stimulation but, instead, may reflect infant reactivity to these novel procedures and setting.

As predicted by the Polyvagal Theory (Porges, 1996), NE infants showed RSA suppression during tasks involving environmental challenge which is indicative of adaptive autonomic nervous system regulation for affect eliciting tasks and is consistent with earlier reports of decreased RSA during arm restraint and other tasks designed to elicit frustration (Stifter & Fox, 1990; Stifter & Jain, 1996). Thus, consistent with the Polyvagal Theory, these infants inhibited the vagal brake. Consistent with our hypothesis and with the pattern of physiological regulation displayed among a sample of 7-month-old CE infants from the present longitudinal study (Schuetze et al., 2007), exposed infants did not display RSA suppression during the NA task. The failure to suppress RSA in response to environmental challenge is consistent with previous findings that infants with regulatory disorders did not regulate (suppress) RSA from baseline to a task (DeGangi et al., 1991) and that this pattern of dysregulation is a long-term effect associated with PCE that is not limited to early infancy. Since other studies have indicated that there is a possible association between vagal regulation and both concurrent and future behavioral regulation during frustration (Calkins, 1997), and social tasks (Doussard-Roosevelt et al., 1997; Suess & Bornstein, 2000), the findings of the current study suggest that CE infants may be at risk for less adaptive behavioral regulation during environmental challenge. However, another possible interpretation is that infants who did not show RSA suppression during the NA task were not distressed and therefore had no need to regulate. Future studies should obtain physiological measures of sympathetic reactivity, such as skin conductance, to further explore this issue.

It is important to note that there were no significant group differences in the change of RSA from baseline to the PA task. One explanation for this is that elicitation of PA may not require active coping and, consequently, RSA suppression is not needed. In fact, some studies have found increases in RSA during PA tasks (Bazhenova, Plonskaia, & Porges, 2001) while others have found that behavioral indicators of PA did not reliably correspond with changes in RSA (Weinberg & Tronick, 1996). Alternatively, our PA task may have elicited a range of emotional responses which may correspond with differential physiological responding. For example, focused attention has been found to correspond with increases in RSA rather than RSA suppression (DiPietro, Porges, and Uhly, 1992).

These findings also indicate that the association between PCE and failure to physiologically regulate during an NA-eliciting procedure is not mediated by MDA. However, MDA did moderate this association such that CE infants who had mothers with high MDA had a larger increase in their RSA during the NA task than CE infants whose mothers had low MDA. NE infants, however, displayed RSA suppression during the NA task regardless of the level of their caregiver's NA, although the suppression was significantly larger for the NE infants whose mothers had low MDA. This provides empirical evidence supporting one specific mechanism by which CE may impact physiological regulation during infancy. These findings can be considered within the context of a vulnerability model which indicates that experiencing aspects of nonoptimal caregiving such as higher levels of MDA in the presence of substance exposure increases developmental vulnerability (Luthar & Zelazo, 2003). The implications for this are underscored by the fact that there is evidence for a gene–environment interaction for RSA suppression during challenge in infants (see Propper et al.,

2008). The same gene that has been linked to RSA suppression during environmental challenge is associated with substance use. Substance-using mothers and their infants may share a genetic profile that manifests itself as physiological dysregulation in their infants. Furthermore, parental characteristics have been shown to mediate this association such that more optimal parental functioning (lower MDA in our study) is associated with less physiological dysregulation in an NE population (Propper et al., 2008). Alternatively, there may be a bidirectional association between MDA and infant regulation. Mothers who have children who are physiologically dysregulated may be more likely to experience symptoms of MDA as a result of increased parenting stress or challenge.

Finally, the present findings provide additional evidence that boys are more vulnerable than girls to the effects of PCE as early as infancy. Although PCE was associated with reduced RSA suppression during tasks designed to elicit both PA and NA among both boys and girls, the effects were exacerbated for exposed boys, suggesting an enhanced vulnerability to the effects of CE for boys. This finding is consistent with the general developmental literature which indicates that boys tend to have less optimal behavioral regulation such as higher levels of NA and more difficulty regulating their emotions than girls (Carter, Mayes, & Pajer, 1990). Furthermore, recent studies have suggested that regulatory behaviors are particularly vulnerable to prenatal cocaine exposure. For example, CE boys, but not girls, were almost twice as likely to have clinically significant levels of externalizing problems (Delaney-Black et al., 2000) and CE boys receive higher ratings of aggression than girls or NE boys (Bendersky et al., 2006). The present findings contribute to this body of knowledge by indicating that gender differences also exist in physiological responsivity to environmental challenge. Longitudinal research is needed, however, to examine whether this gender difference for less optimal physiological regulation persists throughout infancy and into the childhood years.

It is important to note two limitations of this study. First, our measure of MDA is based on maternal self-report. Consequently, women may have misrepresented their levels of symptomatology. However, it is noteworthy that there are significant group differences for MDA and this domain is associated with observed physiological indices of regulation among the infants. Second, although care was taken in the present study to identify substance use in this sample, the accurate assessment of substance use is always difficult, particularly among pregnant women. Pregnant women are often hesitant to divulge information regarding the use of substances during pregnancy, particularly if illicit substances such as cocaine. To address this issue, multiple indices of substance use were used, including self-report using the reliable TLFB interview as well as analysis of medical records, and hair and urine samples. Each of these measures has its own limitations. However, when used in combination, they greatly increase the likelihood of accurately identifying peripartum substance use.

Despite these limitations, the present findings are important because they provide additional support for the influence of PCE on infant regulation. In particular, they extend findings of an association between PCE and physiological regulation into later infancy. The importance of these findings is underscored by the large developmental literature suggesting that this pattern of physiological dysregulation is linked to a wide range of subsequent developmental problems. Furthermore, these findings suggest that there are multiple pathways to risk among CE infants and that it is important to consider both infant and caregiver characteristics as additional influences on the development of regulatory processes in this population.

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Abbreviations

PCE	cocaine exposure
CE	cocaine exposed
NE	nonexposed
MDA	maternal depression or anxiety
NA	negative affect
PA	positive affect
RSA	respiratory sinus arrhythmia
RSA-PA	change in RSA from baseline to a positive affect task
RSA-NA	change in RSA from baseline to negative affect task
BRSA	baseline RSA

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Figure 1.
Group differences in respiratory sinus arrhythmia across epochs

Figure 2.
Interaction effect for gender and cocaine exposure status on RSA regulation during a PA task

Figure 3. Interaction effect for gender and cocaine exposure status on RSA regulation during an NA task

Figure 4.
Interaction effects for MDA and cocaine exposure status on RSA regulation during an NA task

Table 1

Group differences for level of cocaine exposure

	Abstainers (n = 75)		Users (n = 79)	
	M	SD	M	SD
Maternal characteristics				
Age (years)**	28.41	5.60	30.93	6.06
Parity*	3.32	1.67	4.11	2.36
Socioeconomic status**	3.65	1.72	2.93	1.43
Maternal education (years)	12.15	1.80	11.82	1.60
Prenatal # of days cocaine/week**	0	0	.70	1.38
Prenatal # of cigarettes/week**	8.92	19.18	38.70	44.17
Prenatal # of standard drinks/week**	.14	.65	5.37	13.72
Prenatal # of binge drinking episodes/week**	.02	.10	.48	1.39
Prenatal # of joints/week	1.13	6.24	1.08	2.69
Postnatal # of days cocaine/week ⁺	.00	.00	.11	.52
Postnatal # of cigarettes/week**	16.49	31.97	38.62	46.28
Postnatal # of standard drinks/week*	.60	1.40	2.04	4.95
Postnatal # of joints/week	.44	2.13	.13	.04
Maternal Depression/Anxiety*	2.34	.55	2.53	.70
Infant characteristics				
Gestational Age**	39.32	1.20	38.62	1.87
Birthweight (grams)**	3328.72	500.49	2978.51	573.47
Birthlength (cm) ⁺	49.68	2.97	48.07	3.31
Head Circumference (cm) at birth	33.52	1.33	33.32	2.37
OCS**	100.37	18.92	85.89	15.93

Notes: High scores on OCS reflect more optimal obstetric scores. Attempts were made to group match for maternal age but because mothers who use cocaine during pregnancy are consistently older, we were not completely successful at full matching for this variable.

⁺ $p < .10$;

* $p < .05$;

$p < .10$
**

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